

TO : Director, NIEHS

DATE: March 1, 1977

FROM : Acting Associate Director
Research Resources ProgramSUBJECT: Studies on Possible Pentachlorophenol/Dibenzodioxin Intoxication
(Progress Report)

The National Institute of Environmental Health Sciences (Dr. Moore) was contacted by Mr. George LeMunyan in December 1976. Mr. LeMunyan owns a dairy farm in Cedar Springs, Michigan. His farm was one which lost all of its cattle during the Michigan PCB incident a couple of years ago. In addition, his farm buildings had burned soon after this incident. He constructed new barn facilities and repopulated his farm with dairy cattle from another state. For several months after this repopulation, his operation from a herd health standpoint was fairly normal. After these first few months, however, he started experiencing various types of problems which included calf loss, illness and death in adult animals, and loss of milk production. These losses continued through 1976. Late in 1976, several animals from the LeMunyan herd were transported to the Veterinary Clinic of Michigan State University for surveillance and evaluation. Tissues from one of these animals collected at the time of necropsy were evaluated for the presence of a number of chemical toxicants. The results of this evaluation revealed the presence of pentachlorophenol (PCP) in the parts-per-billion range. Mr. LeMunyan contacted me to see if I could offer any suggestions or assistance. I discussed this farm's problem with Dr. David Ellis of Michigan State University with a focus not on pentachlorophenol intoxication per se, but an intoxication that could be a consequence of chemical contaminants associated with PCP; i.e., dibenzodioxins and dibenzofurans.

In early January 1977, while in Lansing, Michigan, to attend a PCB meeting, I traveled to the LeMunyan farm with Drs. Ellis and Davis (Michigan Dept of Agriculture). It was agreed that one had to determine if the PCP exposure found in the one animal was an isolated instance or a generalized circumstance. To this end, we bled a number of ill animals. HSU had the blood samples evaluated for the presence of pentachlorophenol, which was found in most every instance. The level of PCP detected is not of sufficient magnitude to be of prime toxicologic concern; it did indicate continual and chronic exposure to PCP (through treated lumber in the new barn). The chronic exposure underscores the potential for accumulation of dibenzodioxins and dibenzofurans which could be of toxicologic significance. I agreed to have tissues from animals evaluated for the presence of dibenzodioxin at NIEHS. Tissues (liver and fat)

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were sent to NIEHS as was a piece of treated lumber from the barn. Dibenzodioxins can only be detected through GC/MS methodologies, an analytical method clearly within our capabilities and experience. We observed the presence of octa-CDD and hepta-CDD in the wood sample. Analyses of liver tissues (after extensive efforts to develop an effective extraction procedure suitable for the higher chlorinated dibenzodioxins) confirmed the presence of dibenzodioxins in 15 of 15 livers. Octa-dioxin values are in the parts-per-billion range with decreasing incidence and levels of heptadioxins and three instances of hexadioxins in the parts-per-trillion range. These analyses are not yet complete.

In my opinion, the presence of these dibenzodioxins (and likely equivalent amounts of the toxic dibenzofurans, although we have not assayed for these) is of prime toxicologic importance. Previous work at NIEHS has shown that the hexa-CDD present (i.e., 1,2,3,7,8,9) is a most toxic dibenzodioxin isomer with a single oral LD50 in guinea-pigs of less than 100 µg/kg. Although the heptachlorinated dibenzodioxin present (1,2,3,4,6,7,8) has not received thorough toxicologic evaluation, its structure is such that one would predict it to have significant toxicologic properties. The octachlorinated dibenzodioxins have been reported by many to be of little or diminished toxicologic significance. This is based in large part upon its extremely poor solubility and consequent lack of absorption. Studies with this compound are all seriously flawed in one way or another. The chronic toxicity of octa-CDD has not been properly evaluated. The "poor" absorption and therefore diminished toxicity cannot be reassuring in this circumstance since we are detecting it in liver. Its possible toxicity cannot be ignored.

Early attempts to analyze fat for these compounds has been unsatisfactory. Most of the "fat samples" failed to yield much extractable lipid indicating that the majority of tissue submitted is of connective tissue origin (this reflects possible cachexia). Where sufficient lipid was extracted, early analyses suggest that dibenzodioxin levels in this tissue may be one to two magnitudes greater than that found in liver.

One of the high values detected was from a young calf liver which, I believe, had been allowed to drink milk produced on the farm. Previous work at NIEHS with 10CDD showed this chemical to be readily secreted in the milk. One must consider the possibility that the excessive calf mortality experienced at the Lehigh farm may be due to the dibenzodioxins received in milk.

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This work is in my opinion the first confirmed instance of the presence of dibenzodioxins in animal tissues as a likely consequence of chronic exposure to PCP treated wood. It has been mentioned to me by a number of practicing veterinarians that this circumstance, although clearly unusual in its severity, may not be a unique situation.

The public health implications are somewhat obvious:

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1. Possible population exposure to dibenzodioxin and dibenzofuran contaminants through use of PCP or treated lumber.
2. Possible presence of these toxic contaminants in commercial milk.
3. The presence of these impurities in meat products.

Note

The Lehighen herd is one which has had extensive press exposure. These results will receive wide coverage in the media.

John A. Moore, D.V.M.

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